APPENDIX A

The numbers assigned to the following article summaries correspond with the numbers assigned to the synopses of the articles in the text of this Report.

LUNG CANCER

[36] **"Previous Cancer and Radiotherapy as Risk Factors for Lung Cancer in Lifetime Nonsmokers," G.C. Kabat, *Cancer Causes and Control* 4: 489-495, 1993**

"Two factors which have received little attention in previous epidemiologic studies are previous primary cancers and history of radiotherapy. The present analysis was undertaken to examine the association of these two variables as risk factors for lung cancer occurring in lifetime nonsmokers."

"All subjects were interviewed in the hospital by trained interviewers who administered a questionnaire covering: demographics; lifetime smoking history; alcohol intake; occupation and occupational exposures; and a detailed history of exposure to environmental tobacco smoke."

"In females, eight out of 47 cases (17 percent) compared with eight out of 132 controls (six percent) reported a prior primary cancer (crude odds ratio [OR] = 3.2, 95 percent confidence interval [CI] = 1.1-9.0). Seven female cases (15 percent) and six controls (five percent) reported having received radiotherapy: crude OR = 3.7 (CI = 1.2-10.9).

"Seven female cases (15 percent) and five controls (four percent) had had a prior primary of a reproductive cancer: OR adjusted for age, years of education, hospital, lifetime environmental tobacco-smoke exposure, and BMI [body mass index] was 4.9 (CI = 1.4-17.7) The OR for history of radiation therapy adjusted for the same variables was 4.4 (CI = 1.3-15.1). When history of radiotherapy and previous reproductive primary were entered simultaneously in the model, the ORs were reduced to 2.9 (CI = 0.7-12.7) for previous reproductive primary, and to 2.2 (CI = 0.5-9.2) for history of radiotherapy."

"The finding in this study of an association of a history of reproductive cancer and a history of radiotherapy with lung cancer in female nonsmokers is of interest in view of the magnitude of the associations (adjusted ORs for either factor entered separately in the model are in the range of 4.0 to 5.0). In comparison, the average relative risk of lung cancer in nonsmoking women with husbands who smoke is 1.34 (CI = 1.18-1.53)."

"Radiation treatment and a history of a previous reproductive cancer were correlated so highly among the cases in these data that it was not possible to examine the effect of one factor independent of the other....The observed interaction between history of radiotherapy and previous reproductive primary suggest that the two variables taken together are better predictors of lung cancer in never-smokers than either alone."

"The associations of a history of reproductive cancer and of a history of radiotherapy with subsequent lung cancer in women can be explained in three ways: (i) the second 'primary' is not a true primary but is due to spread of the reproductive cancer; (ii) radiation treatment for a prior condition induced the lung cancers; and (iii) lung cancer in women may share certain nontobacco risk actors (such as reproductive factors) with breast, endometrial, and other reproductive cancers."

"Limitations of the present study include: the small number of subjects with a previous primary or a history of radiotherapy; the combining of cancers of different sites; and the lack of an independent review of lung cancer histology. Larger datasets will be required to evaluate the association of specific previous primaries and history of radiotherapy with subsequent lung cancer in women."

[37] **"Incidence of Cancer Among Male Waiters and Cooks: Two Norwegian Cohorts," K. Kjaerheim and A. Andersen, *Cancer Causes and Control* 4: 419-426, 1993**

"The consumption of alcohol varies in different occupational groups, and occupations of waiter as well as cook have been identified at high risk for high alcohol consumption and alcoholism. The occupation of waiter has been found to be among those with the highest percentage of daily smokers, and, additionally, waiters are exposed to passive smoking at work, which also has been associated with increased risk of lung cancer."

"Because few cohort studies have been performed on cancer incidence in restaurant workers, which are thought to be high risk groups for cancer, we decided to analyze cancer incidence in two historical cohorts of male waiters and cooks."

"The present study shows an excess risk of alcohol-associated cancer cases among waiters and cooks alike, and an excess of lung cancer cases among waiters only. The division of the cohorts into persons living in Oslo and persons living outside Oslo showed very similar results for the two residential subgroups. This indicates that the elevations of the alcohol- and tobacco-related cancers in these cohorts are not an exclusively big-city phenomenon, but are linked to the occupational status as such."

"The main results suggest the most important etiologic factors to be alcohol consumption or tobacco smoking, alone or in combination. It is a limitation of this study that individual information on exposure to alcohol and tobacco is lacking."

"Taking into account the partly different working environments of the two groups, passive smoking also should be considered as a possible explanation of the difference in lung cancer ratio. Research on passive smoking in association with lung cancer has shown risk ratios ranging between 1.24 and 1.44, but most of this research refers to persons with a much lower level of exposure than waiters. Few, if any, occupational groups are exposed to passive smoking in the way waiters are. Studies of cotinine in sputum from non-smoking bartenders have shown median cotinine levels of 7.95 ng/ml, with maximum levels exceeding 30 ng/ml. For purposes of comparison, levels between 1.35 ng/ml and 2.15 ng/ml have been found in 11- to 16-year-old children when one parent smokes. It also has been shown that an increased level of blood cotinine corresponds with increased levels of carcinogens in blood."

"This study has shown an excess of cancer cases associated with alcohol consumptions and tobacco smoking in waiters, and with alcohol consumption in cooks. Comparison of the present results with the results of other studies and with our own exposure data tend to indicate that alcohol and tobacco are the main hazards. However, whether these results are due to selective forces in the recruitment of persons to the occupations of waiter and cook, the easy availability of alcohol, coping strategies for stress factors in the work environment, or a cultural phenomenon of drinking and smoking habits in this group, are questions to be discussed in later research."

Other Health Issues

[38] "Complex Mixtures of Tobacco Smoke and the Occupational Environment," D.M. Aviado. In: *Patty's Industrial Hygiene and Toxicology* (Fourth Edition, Volume 2, Part A). G.D. Clayton and F.E. Clayton (eds.). John Wiley and Sons, 107-148, 1993

"The scientific question is as follows: Is nonoccupational or household exposure to chemicals relevant to workplace exposure? The question applies to recent events relating to possible health effects of worker exposure to environmental tobacco smoke (ETS)."

"The potential association of increased disease risk with ETS exposure to nonsmoking workers is presently being considered by OSHA. According to a Current Intelligence Bulletin released in 1991 by NIOSH, there is an association between spousal smoking and increased incidence of certain cardiopulmonary diseases, especially lung cancer, but possibly heart disease as well. The association was derived from epidemiologic studies of nonsmokers who reported exposure from cigarette smoking spouses. So far, there are no reported epidemiologic studies designed to examine the incidence of cardiopulmonary disease in nonsmoking workers sharing work facilities with smoking workers. Only respiratory tract irritation has been reported, but there are no specific studies on occupational heart disease, neoplastic disease, and respiratory tract disease."

"Respiratory tract irritation has been reported to be associated with constituents in ETS. Workers in crowded and smoke-filled bars, restaurants, kitchens, and other public places report episodes of mucosal irritation of the nose, mouth, throat, respiratory airways, and conjunctivae. The reported elevated levels of nicotine and other constituents in ETS are less than the respective TLVs, suggesting that any respiratory tract complaints are the summation of subthreshold effects. Any one of a dozen constituents can be used as

a marker for potential irritants in ETS, but the most informative is the specific prototype, that is, nicotine. Nonventilated chambers have been used to simulate smoke-filled rooms, and the phenomenon of respiratory tract irritation seen in workplaces has been reported. The irritation is readily reversible by resumption of adequate ventilation to dispose of high levels of ETS."

"The potential associations between spousal smoking and the incidence of cardiopulmonary disease cannot be extrapolated to workers. Spousal heart disease, spousal neoplastic disease, and childhood bronchial asthma are cardiopulmonary disease entities with clinical features and pathogenesis different from corresponding occupational diseases. Other than the obvious difference in age (childhood versus adult) and gender (spousal disease in women versus occupational disease in men), there are compelling differences. Epidemiologic studies relating to household exposure and spousal and childhood diseases consist almost entirely of questionnaire responses. If there is personal contact between medical personnel and subject (child or spouse), it usually occurs once for the explicit purpose of obtaining past medical history and estimating ETS exposure, but without actual measurement of ETS levels. There has been no uniformity in questionnaires used so that deficiencies of earlier epidemiologic studies are only partially corrected in later ones."

"The evolution of occupational disease is an extended process lasting for several years. Medical records prior to employment and yearly examinations are necessary to detect the occurrence of cardiopulmonary disease. Diagnostic and research techniques for detection of occupational heart disease have been cataloged, together with relevant experimental animal procedures to serve as a model for reviewing whether ETS exposure of workers is associated with any form of heart disease. Because workers not exposed to ETS and workers exposed to ETS in the same occupational group have not been medically followed for several years, there is no justification to apply the reasoning that because home exposure through spousal smoking might be associated with cardiopulmonary disease, then workplace ETS exposure can be expected to have a similar association."

"Results of household exposure cannot be applied to workers. The ongoing controversy between work standards proposed by ACGIH and those proposed by NIOSH is in part the outcome of differences in opinion. Only data derived from occupational groups are used by ACGIH, whereas NIOSH relies on data derived form nonoccupational and family groups. The controversy extends to the consideration of occupational diseases purportedly associated with ETS. Confounding factors related to ETS exposure in workplaces are different from factors influencing spouses and children in studies of household exposure. Dietary factors and household pollution may influence the incidence of spousal and childhood diseases. On the other hand, workers are exposed to industrial chemicals and outdoor pollutants as well as work-related stress. Familial patterns of inherited or acquired susceptibility to cardiopulmonary disease do not apply to occupational groups."

"Unlike household exposure, workplace exposure can be monitored by good industrial hygiene practice. There are adequate ETS markers for exposures to certain constituents: carbon monoxide for ischemic heart disease, benzo[a]pyrene for neoplastic diseases, and nicotine for respiratory tract irritation. These markers can continue to be monitored in a prospective study of workers differentiated according to ETS exposure. Such a study will require considerable expense and human effort. A simple recourse is to regard ETS as a complex mixture, similar to vehicular emission. As long as carbon monoxide is not allowed to accumulate in workplaces by adequate ventilation, ETS is unlikely to be associated with reports of respiratory tract irritation. ETS levels that are sufficiently high to provoke complaints of irritation can serve as an indication that corrective measures should be initiated to increase workplace ventilation."

[39] "Nicotine Exposure and the Risk of SIDS," J. Milerad and H. Sundell, *Acta Paediatrica* (Suppl.)389: 70-72, 1993

"Several epidemiological studies indicate that smoking during pregnancy and nursing is a major and independent risk factor for sudden infant death syndrome (SIDS)."

"Parental smoking also increases morbidity in lower respiratory tract infections, which is another factor associated with SIDS. However, the increased risk remains after correction for socioeconomic factors. The secondary effects of passive smoking are thus not sufficient to explain the increased risk."

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"Passive smoking and breast milk transfer have both been shown to produce significant levels of cotinine in urine and plasma in exposed infants. While victims of SIDS have been exposed to passive smoking more often, it is not clear if there is a relationship between nicotine exposure and death."

"We have investigated if exposure coincides with the time of death by analyzing the levels of nicotine and cotinine in the pericardial fluid in 24 consecutive deaths in children under the age of one year. As is typical of the clinical history of SIDS, the infants had died suddenly without known previous disease. Sixteen cases had no positive findings at autopsy, while contributing factors were found in eight infants. We found that almost half of our cases had been exposed to nicotine (cotinine levels 5-50 ng) and one-third heavily exposed around the time of death (cotinine > 50 ng)."

"Therefore, death from SIDS seems to coincide more often with nicotine exposure than what could be expected from smoking habits in the general population."

"The effects of parental smoking and nicotine exposure link a number of seemingly independent observations in SIDS research. There is accumulating evidence that fetal development and wellbeing are closely related to the subsequent risk of SIDS in the offspring. Furthermore, it appears that this risk is related, in particular, to alterations in brain development. The risk of dying of SIDS may thus, in reality, be confined to a limited number of infants with developmental changes in CNS function. Our findings of age-specific attenuation of hypoxic defense following nicotine exposure focus the attention on brain catecholamine metabolism as a potential target for adverse fetal and neonatal influences. To clarify the mechanisms of nicotine exposure on postnatal development of the control of respiration and cardiovascular control may thus offer insights into the ultimate mechanism of SIDS."

ETS Exposure and Monitoring

[40] "Relevance of Nicotine Content of Common Vegetables to the Identification of Passive Tobacco Smokers," E.F. Domino, E. Hornbach, and T. Demana, *Medical Science Research* 21: 571-572, 1993

"It is a common experience when studying the blood and urine levels of nicotine and its metabolites in tobacco smokers compared with nonsmokers to find that many of the latter have small but detectable levels of nicotine and its metabolites....The presence of nicotine and cotinine in body fluids of nonsmokers is usually taken as evidence of passive smoking exposure. This is because nicotine when present in air is almost always due to contamination by tobacco smoke."

"Very recently, the Center[s] for Disease Control and Prevention completed a study of 800 people, both smokers and nonsmokers, who all tested positive for cotinine. It has been our experience that some nonsmokers in whom small amounts of nicotine and cotinine were present insist that they had absolutely no contact with tobacco smoke and smokers for extended periods of time sufficient that nicotine and its metabolites should have been excreted."

"Even when precise nicotine-fee analytical techniques are used to insure that no nicotine contamination has occurred, some body fluids of nonsmokers not exposed to environmental tobacco smoke still contain small amounts of nicotine and cotinine. Dietary sources of nicotine in common foods could be responsible for the nicotine and cotinine concentrations in the body fluids of some nonsmokers."

"There has been considerable evidence over the years that nicotine is present in certain foods, especially plants from the family Solanaceae."

"The purpose of the present research was to confirm these reports in our own laboratory...as the basis of a future possible human study on the actual ingestion of nicotine containing foods in nonsmokers."

"If one assumes complete absorption of nicotine from the lungs, then it would take about 142-238 min of breathing air from a minimal smoke environment or the ingestion of about 140 g of cooked potatoes to achieve equal body exposure to nicotine assuming 100% oral nicotine bioavailability."

"One pound of potatoes is approximately 455 g or about 2 medium sized potatoes. The nonsmoker would need to be in a low concentration smoky room for 455/140 X 143 to 238 = 465 to 774 min to obtain the equivalent amount of nicotine to eating about one pound of medium sized potatoes available from one particular supermarket in Ann Arbor, MI."

"Three additional points should be made regarding nicotine exposure and its urinary metabolites."

"(1) Nicotine biotransformation in humans is highly variable."

"(2) Nicotine in environmental tobacco smoke is in a vapour phase that readily adheres to many surfaces. One may be exposed to nicotine as it desorbs from such surfaces."

"(3) The US Environmental Protection Agency has assumed that control groups in epidemiologic studies are exposed to environmental tobacco smoke based on the presence of cotinine in the control group's urine. As a result, the Environmental Protection Agency uses a background correction that raises the risk estimate from 1.19 to 1.59. Obviously, low levels of cotinine in urine can occur from other sources than tobacco smoking, making a background correction erroneous. The above issues have been discussed previously by others but need to be re-emphasized."

"It appears that the dietary intake of nicotine in nonsmokers is of practical importance in the interpretation of passive smoke inhalation by nonsmokers when determining blood and urinary nicotine and cotinine levels. Further research correlating the amount of vegetable intake with urine levels of nicotine and its metabolites is necessary and should be pursued."

INDOOR AIR QUALITY

[41] "Is It Time to Change the Terminology of Sick Building Syndrome?" B. Jarvholm, *Indoor Environment* 2: 186-188, 1993

"'Sick Building Syndrome' (SBS) is a common term in indoor environment literature. . . . Over the years, SBS has been used to describe many symptoms which are believed to be caused by some factor(s) in buildings....The custom of using SBS for a broad range of symptoms, usually of unknown origin, has made the term less useful. Is it time to abandon SBS and use a new and more distinct terminology?"

"There are several reasons why the use of the term SBS should be avoided."

"(1) SBS is vaguely defined. It consists usually of some symptoms related to a nonindustrial workplace. However, it is unclear how many of the symptoms should occur and if they are and how they should be associated with the building. Some authors obviously use the term without the knowledge of whether or not the symptoms are causally linked to the building, e.g., its walls, floors or ventilation system."

"(2) The term SBS suggests that there is knowledge that the symptoms actually are caused by factors in the building, while the term usually is used in situations where such knowledge is missing."

"(3) It is sometimes claimed that SBS is a multifactorial disease. Such information is of little value, as most or all diseases have multifactorial causes."

"(4) Medical diagnoses and syndromes are usually based on pathological changes in certain organs . . . in other cases, they are based on etiological factors...SBS does not satisfy any of these criteria."

"(5) It is now at least 10 years since SBS was defined by a WHO working group. The term has obviously not improved the understanding of the occurrence of these symptoms."

"If it can be shown that the illness is caused by some factor(s) related to the ventilation system, it must be much better to report that, for example, 'there is an increased prevalence of mucous membrane irritation and lethargy due to malfunction of the ventilation system' than to say that 'there is a SBS'. Thus, it is suggested that the medical problem should be described in ordinary terms."

[42] "New Approaches for the Determination of Ventilation Rates: The Role of Sensory Perception," M.V. Jokl, G.B. Leslie, and L.S. Levy, *Indoor Environment* 2: 143-148, 1993

"The Commission of the European Communities recently issued a report proposing 'Guidelines for Ventilation Requirements in Buildings'. This report makes recommendations on appropriate ventilating rates for buildings in order both to minimize any health risk for

building occupants and to ensure that the air is perceived as fresh and comfortable by the occupants."

"The guidelines suggest that perceived sensory pollution can be used to determine necessary ventilation rates during the design stage of a building."

"An existing alternative to the guidelines proposed in the EEC report is the American Society of Hearing, Refrigerating and Air-Conditioning Engineers (ASHRAE) Standard 62-1989."

"This paper compares the ASHRAE approach with that suggested by the EEC. More specifically, it assesses the use of decipol/olf values as the parameters for defining ventilation rates in European buildings."

"Because of the diversity of the sources and types of indoor air pollutants, many have advocated that the best way in which to ensure good indoor air quality is to provide sufficient ventilation. In this manner the accumulation of indoor air pollutants can be avoided by adequate replacement of stale air by fresh air. This approach can provide comfort and protect health."

"The most recent guideline from ASHRAE on indoor air quality management is a standard broadly based on the provision of sufficient ventilation."

"The EEC guidelines also aim to ensure adequate indoor air quality from the standpoint of both health and comfort for the occupants."

"The guidelines suggest that, in practice, comfort rather than health defines the required ventilation. The comfort criteria depend on the use of the olf concept. Because typically odour is sensed before irritation or other subjective sensory reaction, this in reality becomes an odour standard."

"One olf unit is defined as the pollution emitted by one standard person, that being an average sedentary adult office worker feeling comfortable with the ambient temperature and with a hygienic standard equivalent to 0.7 baths per day."

"On initial consideration it may seem that the concept of using sensory perception as the determinant of air quality is a helpful and sensible approach. However, the manner in which this is applied to the EEC guidelines for setting ventilation rates for air-conditioned buildings seems premature and has practical difficulties. For one, it leaves far too much to guesswork. Many of the parameters necessary to use this approach are not known, or at least are not defined fully. Even if there were a larger database on the sensory pollution loads associated with buildings, odour is not necessarily the proper marker for maintaining the health of the building occupants and may, in some circumstances, underestimate the risks to health. The EEC report does have a provision to estimate ventilation rates based on health considerations, but again far too few of the parameters involved in this estimation are known to allow an architect to design a building based on these guidelines. To assist in this, the WHO Air Quality Guidelines for Europe are summarised in an appendix, but are not really incorporated into the ventilation guidelines for the European Community."

"A far more practical approach is given in the ASHRAE Standard 62-1989. Here minimum ventilation rates for various environments are defined, so that the designer has a specific and fixed target to achieve. This standard has been in operation in the United States for a number of years, and used in many other countries. It seems to work well. Clearly, it has its faults but it is simple, practical and relies little on assumptions which may or may not be the prime determinants of both comfort and health risk. Whether precisely this standard is the best one for the European Community is open to debate, but the general concept employed by ASHRAE at least points in a direction which can be achieved. Sensory perception of occupants is, of course, an important consideration in ventilation design, but the way in which it is incorporated into guidelines for ventilation calculations seems to have little relevance to health."

[43] "Status of ASHRAE Standard 62 -- Ventilation for Acceptable Indoor Air Quality," W.G. Tucker, *Proceedings of Indoor Air '93* 3: 525-530, 1993

"This paper briefly describes the purpose, history, and major features of American Society of Heating, Refrigerating, and Air-conditioning Engineers (ASHRAE) Standard 62. The primary focus of the paper is on the status of the review and revision process as of January 1993. The current working outline of the revised standard is presented and discussed. The complementary roles of ventilation, source management, and air cleaning are emphasized. Extending the standard beyond ventilation system design to include operation

and maintenance of buildings and ventilation systems is also projected."

"As part of ASHRAE's policy to review standards every 5 years, a new committee was formed in January 1992 to review Standard 62-1989, and to develop revisions as needed. The committee consists of 25 designers, equipment manufacturers, regulators, and researchers."

"Special emphasis is being placed on reviewing the scientific information that can be used as the basis for the health and sensory comfort guidance that the document uses to define acceptable indoor air quality. Another special emphasis is on making clearer links between sources of contaminants and control of indoor air quality by ventilation, source management, or air cleaning. Progress in these two areas is key to improving the soundness and usefulness of the two design procedures."

"The current review is focused on issues that have been raised over the past several years in various technical meetings and in feedback to ASHRAE from the design community. It is unreasonable to expect ventilation, which acts primarily by dilution and displacement of indoor contaminants, to provide acceptable indoor air quality by itself. Special emphasis is therefore being given to the health and comfort basis of the standard and to describing the complementary roles of source control and air cleaning for providing good indoor air quality. The review committee is also committed to simplifying and clarifying the standard where possible, in response to requests from design engineers and building code officials."

"The primary changes from Standard 62-1989 that are currently being considered are to provide separate sections for residential buildings and design documentation; new material on operations and maintenance and design calculations; more explicit treatment of source management and air cleaning as options to ventilation for ensuring acceptable indoor air quality; and clarifications or modifications to the Ventilation Rate and Indoor Air Quality Procedures. It is also the revision committee's intention to use 'code language' to make Standard 62 easier to adopt by governmental bodies that establish legal specifications (codes) regarding design and operation of buildings and their HVAC systems. An alterative may be to prepare a specification version for code officials, and a more extensive version with guidance for designers and building owners and operators."

"Other ventilation standards and guidelines are being reviewed for ideas on how Standard 62 should be structured. Under the European ventilation guidelines, three design levels are offered for perceived indoor air quality (which is similar to sensory comfort, as used in this paper). Ventilation rates are calculated separately for health and perceived air quality; the higher rate is recommended for design. These and other approaches will be considered by the SSPC62 committee."

APPENDIX B

UPCOMING SCIENTIFIC MEETINGS

- **November 7-10, 1993**
  Indoor Air Quality '93: Operating and Maintaining Buildings for Health, Comfort and Productivity, ASHRAE, Philadelphia, Pennsylvania [Issue 49, Item 34]
- **November 11-13, 1993**
  Contemporary Concepts of Indoor Air Quality, Hacettepe University, Ankara, Turkey [Issue 49, Item 36]
- **November 16-17, 1993**
  Bugs, Mold & Rot II: Control of Humidity for Health, Artifacts and Buildings, Building Environment and Thermal Envelope Council, Alexandria, Virginia [In This Issue]
- **December 15, 1993**
  Indoor Air Quality: An Overview for People Who Need to Know, AIHHM, San Antonio, Texas [Issue 57, Item 35] Same program to be held March 4, 1994, Orlando, Florida; April 13, 1994, Minneapolis, Minnesota; May 5, 1994, Chicago, Illinois; June 17, 1994, Oklahoma City, Oklahoma; July 14, 1994, Anchorage, Alaska
- **December 16-17, 1993**
  The National Environmental Tobacco Smoke Conference: Public Battles, Private Choices, IAQ Publications, Washington, D.C. [Issue 55, Item 34]
- **March 28-31, 1994**
  Eleventh ORNL Life Sciences Symposium, Indoor Air and Human Health Revisited (Bringing Selected Advances in Medical Science to the Indoor Air Quality Community), Knoxville, Tennessee [Issue 58, Item 43]
- **May 5-7, 1994**
  Second Annual IAQ Conference and Exposition, NCIAQ, Tampa, Florida [Issue 49, Item 35]
- **May 22, 1994**
  Indoor Air Quality Symposium, American Industrial Hygiene Conference and Exposition, Anaheim, California [Issue 57, Item 34]
- **October 18-20, 1994**
  Indoor Air Quality in Asia, Beijing, China [Issue 54, Item 42]
- **October 30-November 2, 1994**
  IAQ '94: Engineering Indoor Environments, ASHRAE and other sponsors, St. Louis, Missouri [Issue 58, Item 42]

ETS/IAQ Report
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